Shortening velocity of human triceps surae muscle measured with the slack test *in vivo*

Kazushige Sasaki and Naokata Ishii

Department of Life Sciences, Graduate School of Arts and Sciences, University of Tokyo, Tokyo 153-8902, Japan

Unloaded shortening velocity (V_0) of human triceps surae muscle was measured *in vivo* by applying the 'slack test', originally developed for determining V_0 of single muscle fibres, to voluntary contractions at varied activation levels (ALs). V_0 was measured from 10 subjects at five different ALs defined as a fraction (5, 10, 20, 40 and 60%) of the maximum voluntary contraction (MVC) torque. Although individual variability was apparent, V_0 tended to increase with AL ($R^2 = 0.089$; $P = 0.035$) up to 60%MVC (8.6 ± 2.6 rad s^{-1}). This value of V_0 at 60%MVC was comparable to the maximum shortening velocity of plantar flexors reported in the previous studies. Electromyographic analysis showed that the activities of soleus, medial gastrocnemius and lateral gastrocnemius muscles increased with AL during isometric contraction and after the application of quick release in a similar manner. Also, it showed that the activity of an antagonist, tibialis anterior muscle, was negligible, even though a slight increase took place after the quick release of agonist. Correlation analysis showed that there were no significant correlations between V_0 and MVC torque normalized with respect to body mass, although the correlation coefficient was relatively high at low ALs. The results suggest that in human muscle, V_0 represents the unloaded velocity of the fastest muscle fibres recruited, and increases with AL possibly because of progressive recruitment of faster fibres. Individual variability may be explained, at least partially, by the difference in fibre-type composition.

(Resubmitted 23 May 2005; accepted after revision 7 July 2005; first published online 7 July 2005)
Corresponding author K. Sasaki: Laboratory of Sports Sciences, Department of Life Sciences, Graduate School of Arts and Sciences, University of Tokyo, Tokyo 153-8902, Japan. Email: cc47710@mail.ecc.u-tokyo.ac.jp

In the locomotory performance of organisms including humans, contractile force and velocity of skeletal muscles play primary roles. Whereas muscle force basically depends on the amount of overlap between actin and myosin filaments in the sarcomere (length–force relation), shortening velocity depends on the ratio between the maximum isometric force and the load applied to the muscle (force–velocity relation). When the load approaches zero, the velocity is ultimately independent of force-generating capacity and depends on the rate of cross-bridge cycling and the number of sarcomeres in series.

Most of the information on contractile force and velocity in human muscles has been obtained by using isokinetic tests, in which joint torque is measured under constant angular velocity. Previous studies have shown the similarity between force–velocity relations obtained from isolated muscles and those from human muscles *in vivo*, i.e. muscle force (or joint torque) decreases progressively as shortening velocity increases. However, a recent study suggests that isokinetic tests can describe the proper force–velocity characteristics of human muscles only in a limited range of velocities (Desplantez & Goubel, 2002). In fact, when using a hyperbola (Hill, 1938) to extrapolate the isokinetic torque–velocity data to the velocity at zero torque (extrapolated maximum velocity; V_{max}), unrealistic values can be found for V_{max} (Desplantez & Goubel, 2002). Therefore, a different approach would be required for appropriate evaluation of the intrinsic shortening velocity in human muscle.

As an alternative to V_{max}, Edman (1979) introduced V_0, the velocity of unloaded shortening determined by the 'slack test'. The slack test involves varied distances of quick release applied to an isometrically contracting muscle fibre. Plotting the time required for the fibre to take up the slack against the distance of release exhibits a linear function, the slope of which provides V_0 of the fibre. This method reveals the unloaded shortening velocity of the contractile component without an effect of the recoil of the series elastic component (SEC) including tendinous tissues, which is independently evaluated by the extrapolation onto the axis of release distance (Edman, 1979).

 DOI: 10.1113/jphysiol.2005.091124

Although the slack test has been widely used for studying contractile properties of single muscle fibres, its application to human muscle *in vivo* is challenging. In voluntary contraction of human muscle, it is necessary to consider that: (1) human limbs, which have a greater inertial mass than that of single muscle fibres, should be moved at a sufficiently high speed; (2) the distance of release must be large due to a large compliance derived from tendons and other soft tissues; (3) synergists and antagonists could simultaneously contribute to the joint torque; and (4) muscle activity would be affected by involuntary reflexes. Thus far, these factors have made the application of the slack test difficult.

In the present study, to overcome these difficulties, we have developed a custom-designed motor-driven dynamometer, with which high angular velocities of up to 20 rad s^{-1} can be attained. The triceps surae, the main plantar flexor muscle of the foot, was chosen as the muscle of interest. This was not only because the inertial mass of the foot is relatively small, but also because the influences of the other plantar flexors and the dorsiflexors on joint torque are negligible (Fukunaga *et al.* 1996; Van Zandwijk *et al.* 1998; Hof, 1998; De Zee & Voigt, 2001). The results suggest that the slack test can be applied to human muscle and unlike the previous results from single muscle fibres (Edman, 1979), V_0 changes with the activation level (AL) of the muscle.

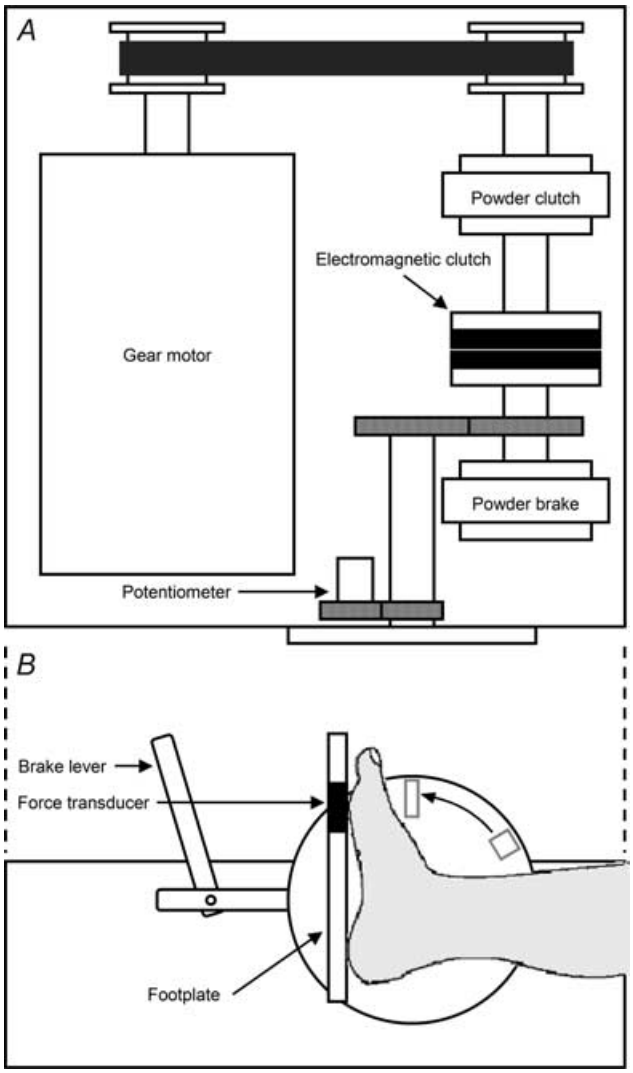

Figure 1. Diagram of the dynamometer for the slack test
Top (*A*) and side (*B*) views are shown. See text for details.

Methods

Subjects

Three women and seven men, who were healthy and had no history of injury affecting the ankle joint, participated in the study. Their mean age, height and body mass were 26.0 ± 5.6 years, 164.7 ± 6.9 cm and 64.1 ± 8.0 kg, respectively. All subjects were informed of the experimental procedure and purpose of this study, which conformed to the standards set by the Declaration of Helsinki, and gave their written consent in accordance with the Ethics Committee for Human Experiments, University of Tokyo.

Equipment

Each subject performed voluntary contractions on the ankle dynamometer, the mechanical set-up of which is shown in Fig. 1. The torque of a gear motor (SG-SMF-08-5, Sigma Giken, Japan) is transmitted to a footplate via an electromagnetic clutch (UC-6, Mitsubishi Electric, Japan). The electromagnetic clutch is engaged by pressing a switch and disengaged when the position of the footplate reaches a certain, adjustable angle determined at 10 deg intervals. Freeing a mechanical brake after engaging the clutch causes a quick-release movement. Two mechanical stops were used to preset the range of angular displacement. The final deceleration and backlash movement of the footplate were damped using a magnetic powder brake (ZKB-1.2XN, Mitsubishi Electric, Japan) and shock-absorbing rubbers attached to the stops. The angular velocity of the footplate was modulated by means of a magnetic powder clutch (ZKB-2.5BN, Mitsubishi Electric, Japan).

The footplate was positioned so that the anatomical axis of the ankle coincided with the axis of the shaft of the dynamometer. Plantar flexion force was measured with a strain-gauge force transducer (LP-B, Kyowa Electronic Instruments, Japan) installed in the footplate so as to make contact with the ball of the foot. Multiplying plantar flexion force by the lever arm length (horizontal distance between the lateral malleolus and the ball of the foot) gives the plantar flexion torque. Angular position was measured with a potentiometer (LP06M3R1HA, Murata

Manufacturing, Japan) attached to the shaft. Both torque and position signals were sampled at 4000 Hz by using a data acquisition system (PowerLab/16SP, ADInstruments, Australia).

In this paper, in accordance with the many previous studies investigating human muscles *in vivo*, muscle forces will be expressed as torques, lengths (distances) as angles, and velocities as angular velocities of the ankle. Assuming that the moment arm length for the Achilles tendon is proportional to the muscle length (Visser *et al.* 1990; Fukunaga *et al.* 2001), the angular velocity of the ankle can be linearly related to the shortening velocity of triceps surae muscle normalized with respect to muscle length.

Testing procedure

A few days or weeks before the testing, all subjects performed one or more orientation sessions with the dynamometer to familiarize themselves with maximum voluntary effort and quick-release movement. During the testing, the subject sat in a chair with the right foot attached firmly to the footplate using three straps. The knee was fully extended and immobilized using two straps.

First, the plantar flexion torque during maximum voluntary contraction (MVC) was measured at an ankle angle of approximately 10 deg dorsiflexion (0 deg represents the angle at which the subject stands erect), achievable without pain or discomfort. Subjects could monitor their own exerted torque on an oscilloscope (SS-7604, Iwatsu Electric, Japan) placed in front of them. Maximum dorsiflexion efforts were also performed at the same ankle angle to normalize the antagonist muscle activation with respect to MVC (see below).

Next, the passive torque from the parallel elasticity and weight of the foot was measured over a wide range of motion, i.e. approximately from 10 deg dorsiflexion to 45 deg plantar flexion. Subjects were asked to relax their leg muscles completely. The footplate was manually rotated at an approximate angular velocity of 0.1 rad s^{-1}.

A series of quick releases was subsequently given at different levels of isometric contraction. The subject could see a target torque as well as the exerted torque on the oscilloscope and keep a steady level of voluntary torque before each release. Both during and after the quick-release movement, the subject was instructed to keep the level of exertion until asked to relax. Activation levels (ALs), defined as target-torque levels during isometric contraction (Phillips & Petrofsky, 1980; Chow & Darling, 1999), were set at 5%, 10%, 20%, 40% and 60% of MVC, and measurements were made in this order. The trials at 5%, 10% and 20% of MVC were repeated three times, whereas those at 40% and 60% of MVC were performed once to minimize the effect of fatigue. For data correction purposes (see below), an additional release was conducted at 0% of MVC (i.e. relaxed condition) after the set of trials at each release distance. Then, the position of the adjustable stop was shifted to change the angular excursion of release, and the same procedure was repeated. The release distance ranged from 25 to 55 deg at intervals of 5 deg. Since a stretch of SEC of the plantar flexors during MVC has been shown to be 0.56 rad (32 deg; Hof, 1998) and 0.3 rad (17 deg; De Zee & Voigt, 2001), it was assumed that the minimum distance of release used in the present study (25–30 deg) was sufficient to slacken the SEC at 5–60% MVC. The effect of passive tension derived from the parallel elastic component was to be considered, because it could accelerate the speed of shortening (Edman, 1979; Claflin *et al.* 1989). However, even the smallest release ended above 10 deg of plantar flexion, where no substantial passive tension was observed (Muir *et al.* 1999).

Finally, the measurements of MVC were made again to test for the presence of fatigue (Chow & Darling, 1999).

Measurement of electromyographic activity

Electromyographic activity (EMG) was recorded throughout the testing session including the passive torque measurements. Bipolar Ag–AgCl surface electrodes (5-mm diameter, 20-mm interelectrode distance) were placed on soleus (SOL), medial gastrocnemius (MG), lateral gastrocnemius (LG), and tibialis anterior (TA) muscles. The reference electrode was placed over the lateral epicondyle of the femur. The skin was shaved, cleaned with alcohol, and abraded to reduce the electrode impedance. All EMG signals were differentially amplified (gain 1000–2000×) with an AC amplifier (AB-610J, Nihon Koden, Japan), band-pass filtered (15–1000 Hz), and sampled at 4000 Hz by using the data acquisition system.

Measurement of foot rotation

It is difficult to completely prevent foot rotation with external strap fixation during 'isometric' plantar flexion (Magnusson *et al.* 2001; Arampatzis *et al.* 2005). Since any foot rotation in the direction of plantar flexion should result in an overestimation of the release distance, an electrical goniometer (XM110/A, Biometrics, UK) was used to monitor the foot rotation during isometric contraction. The end blocks of the goniometer were secured with tape over the medial aspect of the foot and the posterolateral aspect of the tibia. The output signal was sampled at 4000 Hz by using the data acquisition system.

Data analysis

Except for EMG, all the signals from quick-release recordings were smoothed by means of a finite-impulse-response digital filter with a cut-off frequency of 150 Hz.

In MVC trials, plantar flexion torque and rectified EMG signals were averaged over a 1-s period after torque reached a plateau. Routinely, the trials were conducted twice. If the torque values obtained were considerably different (by $>\pm 10\%$), the third trial was conducted. The highest value was determined as MVC, and over the same period the mean rectified EMG was determined as $mEMG_{max}$.

The raw torque signals included considerable inertial artefacts due to high acceleration and deceleration inherent in the quick-release movement. In order to remove these artefacts, the correction method described by De Zee & Voigt (2001, 2002) was used with some modification. Briefly, this method involved the angular acceleration signal (as the second derivative of angular displacement) as well as the two additional recordings mentioned above. The first was the recording of passive torque, which gave a passive torque–angle curve. The second was the recording during the release at the relaxed condition. From this recording, a transfer function H was obtained with the Fourier transform of angular acceleration (input) and the Fourier transform of torque corrected by the passive torque–angle curve (output). The angular acceleration during the release with contraction was Fourier transformed and multiplied by H to estimate the inertial component in the frequency domain, which was subsequently inverse-transformed to the time domain. Finally, the raw torque signal was corrected for the inertial and passive component. Figure 2 shows an example of the data correction.

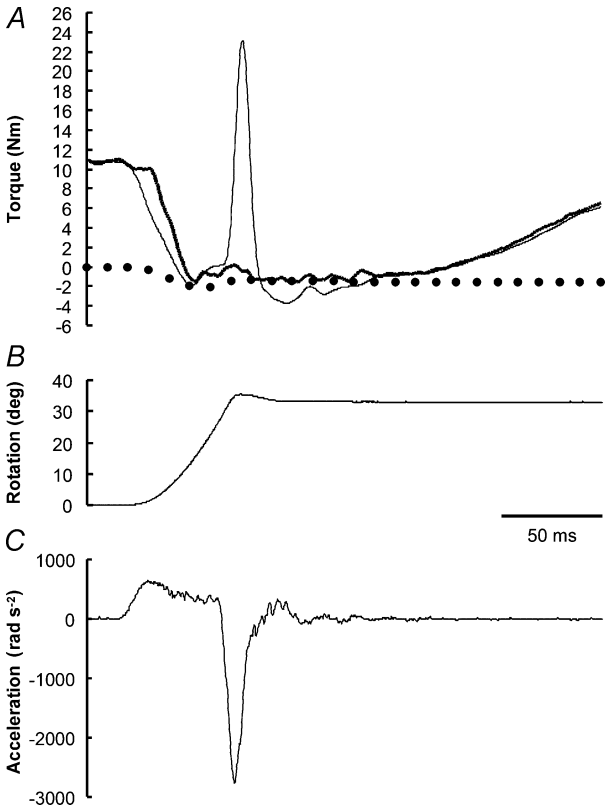

Figure 2. Example of torque (*A*), angle (*B*) and angular acceleration (*C*) recordings during a quick-release movment (subject K, 10%MVC)
The torque recorded by the transducer (thin line) was corrected for inertia effects by using a transfer function (thick line). The passive torque (dotted line) is also presented. Note that the inertial artefact at the end of the release was greatly reduced after correction.

Corrected data were further analysed as shown in Fig. 3. The time, Δt, was measured as the interval between the onset of release (defined as the point at which the angular displacement exceeded 0.3 deg) and the beginning of torque redevelopment. To minimize the effect of noise signals and the uncertainty in the onset of torque redevelopment (Julian *et al.* 1986), the time point at which torque increases above the baseline (i.e. 0 Nm) was calculated from a linear regression fit (Janssen & De Tombe, 1997; Minajeva *et al.* 2002). The torque-redevelopment phase selected was fitted by a linear regression, and its intersection with the torque baseline was determined to be the onset of torque redevelopment. The selection for the fit was repeated twice from a given torque recording, and the mean value was used for further analysis. The coefficient of variation for repeated measurements was 4.4% on average. The distance of release, ΔL, was corrected for the foot rotation averaged over a 100-ms duration just before the release. Relations between Δt and ΔL for varied release distances were fitted to a linear function with least-squares regression. As previously reported (Edman, 1979), the slope of the linear regression provided a measure of the shortening velocity under zero load, V_0.

Electromechanical delay (EMD), the time lag between electromyographic and mechanical activities, should be taken into consideration especially when analysing rapid movements. Published values for EMD, however, have varied greatly and appeared to be affected by several factors such as the initial 'slack' in the SEC at the onset of contraction (Vint *et al.* 2001; Muraoka *et al.* 2004). The results of these studies suggest that adopting a constant EMD value to align EMG with kinetic data is unreasonable. Therefore, in this study the rectified EMG signals were averaged over two different phases of contraction (Fig. 3), a 100-ms period just before the release ($mEMG_a$), and a period between the onset of release and the beginning of torque redevelopment ($mEMG_b$). Comparing EMG activities in static ($mEMG_a$) and dynamic ($mEMG_b$) conditions allowed us to see whether the muscle activity is constant throughout the contraction, regardless of the presence of EMD. The mEMGs obtained were normalized with respect to $mEMG_{max}$ and expressed as a percentage.

Statistics

Data are presented as means and S.D. Statistical analysis was performed with StatView 5.0 (SAS Institute, USA). Regression analysis was used to analyse the effect of AL on the parameters. The least-squares method was used for regression. Analysis of covariance (ANCOVA) was used, when appropriate, to compare the relations between AL (covariate) and mEMGs ($mEMG_a$ and $mEMG_b$). Student's paired t test was used to examine if the MVC differed between the two measurements (before and after the testing session). $P < 0.05$ was considered significant.

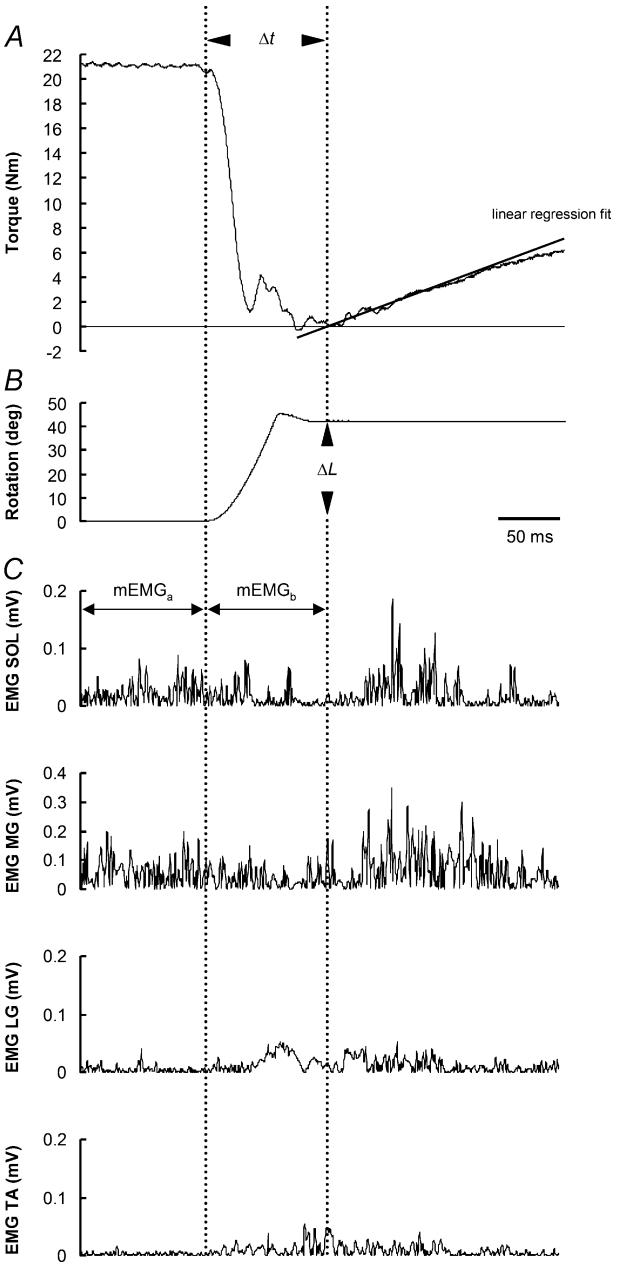

Figure 3. Example of the data analysis (subject K, 20%MVC)
The torque recording (*A*) has been corrected for passive torque and inertial artefact (see Fig. 2). The ripple seen at the end of release is a remainder of inertial artefact. The quick-release distance (ΔL) is expressed as rotation angle of the footplate (*B*). The time, Δt, was measured as the interval between the onset of release and the beginning of torque redevelopment, the latter of which was determined by the linear regression fit. Rectified EMG signals (*C*) were averaged over two different phases of contraction, a 100-ms period just before the release ($mEMG_a$) and a period between the onset of release and the beginning of torque redevelopment ($mEMG_b$).

Results

MVC torque measured after the testing session (93.2 ± 19.5 Nm) was 95.4 ± 10.7% of that measured before the session (97.9 ± 18.7 Nm), and no significant difference was found between the two values ($P = 0.23$). For all subjects, the torque output during isometric contraction matched well the target torque level. The deviations of the torque immediately before the release (averaged over a 100-ms period) from the target torque were 3.4 ± 2.8, 2.3 ± 1.3, 2.2 ± 1.4, 2.1 ± 2.2 and 4.6 ± 3.1% for 5, 10, 20, 40 and 60%MVC trials, respectively.

Figure 4 illustrates superimposed angle and torque recordings from a series of quick releases with different distances. The torque recordings have been corrected for passive torque and inertial artefacts. The quick release given during the plateau of an isometric contraction induced a rapid decrease in torque, and the torque remained constant as the muscle shortened to take up the slack.

Although scattered especially at low AL, the relations between Δt and ΔL could be fitted with a straight line

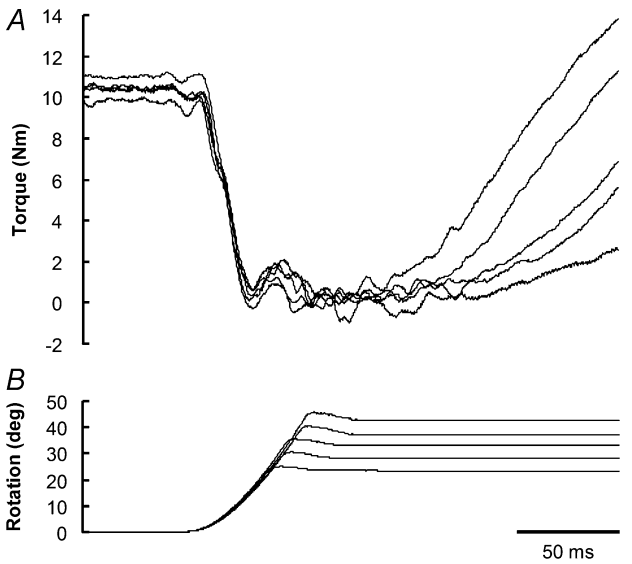

Figure 4. Superimposed torque (*A*) and angle (*B*) recordings from five quick releases with different release distances (subject K, 10%MVC)
The torque recordings have been corrected for passive torque and inertial artefacts (see Fig. 2). The time to torque redevelopment increased with the release distance expressed as rotation angle of the footplate.

($R^2 = 0.50$–0.95). Figure 5 shows examples of the fit from two subjects. The slope of the fitted line, which represents V_0, for subject K (left panel) appeared to be independent of AL, whereas that for subject M (right panel) appeared to increase gradually with AL. Despite these individual variations, the mean value of V_0 significantly increased with AL ($R^2 = 0.089$; $P = 0.035$; Fig. 6). The intercept of the vertical axis by the fitted line also tended to increase with AL. However, the statistical analysis of the intercept was not performed, because the intercept value obtained in the present study does not represent directly an extension of SEC (see Discussion).

Values of mean rectified EMG ($mEMG_a$ and $mEMG_b$) during quick-release trials are shown in Table 1. During isometric contraction just before the release, $mEMG_a$ of SOL, MG, LG and TA significantly increased as AL increased from 5 to 60%MVC ($P < 0.0001$, all muscles). During shortening, $mEMG_b$ of SOL, MG, LG and TA increased with AL in a manner similar to that during isometric contraction ($P < 0.0001$, SOL, MG and LG; $P = 0.0038$, TA). ANCOVA results demonstrated that the activity of TA during shortening was significantly larger than that during isometric contraction ($P < 0.0001$), whereas the agonist activities during shortening were comparable with those during isometric contraction ($P = 0.45$, MG; $P = 0.57$, LG). Since a significant interaction was observed in SOL ($P = 0.0079$), we did not compare mEMGs of SOL.

We found that there were no significant correlations between V_0 and normalized MVC (joint torque per body mass), the latter of which is a measure of muscle quality and thus represents individual force-generating capacity (Sipila *et al.* 1996; Seger & Thorstensson, 2000). The correlation coefficient was higher at low ALs, especially at 10%MVC ($r = 0.54$; $P = 0.11$), whereas V_0 at 60%MVC was almost independent of normalized MVC ($r = -0.076$; $P = 0.84$).

Discussion

The present study aimed to investigate shortening velocities of human triceps surae muscle with the slack test. To our knowledge, no successful study has thus far been reported for the application of the slack test to human muscle contractions *in vivo*. Since there was no difference between the MVC torques determined before and after the testing session, the influence of fatigue was assumed to be negligible.

Determination of unloaded shortening velocity

Although the relations between the time to torque redevelopment (Δt) and the distance of release (ΔL) were fitted to a linear function, the data plotted were somewhat scattered in some cases (Fig. 5), as compared to the reported data for *in vitro* experiments (e.g. Edman, 1979). There are at least two possible reasons for this variation. The first lies in the neural factors associated with the voluntary contraction. In this study, each AL was defined as a fraction of MVC, but it is unlikely that the same population of motor units was consistently activated at each AL. Moreover, the muscle activity during and after the quick release showed fluctuations (as observed in Fig. 3), which could vary from one trial to another. These fluctuations in muscle activity would imply the variable recruitment patterns of motor units during the period of shortening, which possibly influenced the values of Δt.

The second reason is related to the methodology used to determine Δt. Julian *et al.* (1986) demonstrated that determining the onset of force redevelopment

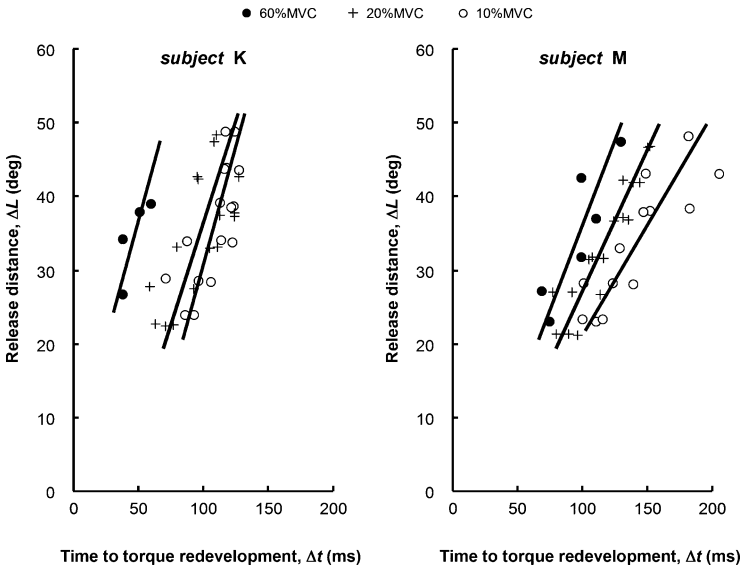

Figure 5. Relations between time to torque redevelopment (Δt) and release distance (ΔL) from subjects K (left panel) and M (right panel)
Only the results from 10, 20 and 60%MVC are shown for clarity. The slopes of linear regression appear to be constant in subject K, whereas those in subject M appear to increase with activation level (AL).

depended on the sensitivity of force recording, and taking the different estimates of Δt resulted in different (approximately 50%) values of V_0. Therefore, in this study the linear regression fit, which is less affected by the sensitivity of recordings, was used and its intersection with the torque baseline was defined as the onset of torque redevelopment. Nonetheless, the torque occasionally fluctuated even in relaxed conditions, which was probably due to slight changes in posture and foot fixation. Even a slight deviation could generate errors, and thus be partly responsible for spreading the values of Δt, which was particularly pertinent at low AL where the absolute rate of torque redevelopment was small.

The relation between unloaded shortening velocity and activation level

The present technique has an advantage in that V_0 of human skeletal muscle can be measured at varied levels of voluntary contraction. Mean rectified EMGs of triceps surae muscle during isometric contraction ($mEMG_a$) and during shortening ($mEMG_b$) were similar, though a significant interaction was found in SOL (Table 1). This was due to the somewhat higher activity at low ALs (5–10% MVC) and the somewhat lower activity at high ALs (40–60%MVC) in $mEMG_b$. Furthermore, the $mEMG_b$ value of TA, which was significantly larger than $mEMG_a$, was still low (ranging from 5.2 to 9.6%$mEMG_{max}$). In an additional experiment on two subjects, we confirmed that such a small amount of TA activity could not cause dorsiflexion even when the activities of agonist were silent, because the magnetic powder brake installed in the dynamometer resisted the rotation. This result suggests that a few per cent of TA activity could not prevent the triceps surae muscle from taking up the slack. Therefore, it is possible to assume that the triceps surae activity remained substantially constant during both isometric and shortening contraction, and that the effect of antagonist coactivation was negligible.

The V_0 value significantly increased with AL (Fig. 6). This appears to be consistent with the previous study for human wrist flexors, in which V_{max} decreased with decreasing activation (Chow & Darling, 1999). It should be noted, however, that V_{max} determined by extrapolation from a force–velocity curve largely depends on the force range used for constructing the curve (Edman, 1979; Josephson & Edman, 1988; Claflin & Faulkner, 1989). Thus, the direct comparison of V_{max} under different levels of voluntary activation would be questionable, unless sufficiently small loads are used to construct the force–velocity curve, which is technically difficult especially at low AL.

On the other hand, V_0 will, in principle, depend upon the shortening velocity of the fastest fibres that have been recruited (Claflin & Faulkner, 1985; Josephson & Edman, 1988). According to the 'size principle' (Henneman *et al.* 1965), small motor units containing slow-twitch fibres (ST) are predominantly activated at low contraction intensities. As contraction intensity increases, larger motor units containing faster muscle fibres are progressively recruited. Therefore, the present data suggest that V_0 would increase from the shortening velocity of ST to that of fast-twitch fibres (FT) with increasing AL. In particular, V_0 at 60%MVC where most of the motor units would be recruited (De Luca *et al.* 1982) is likely to be close to the

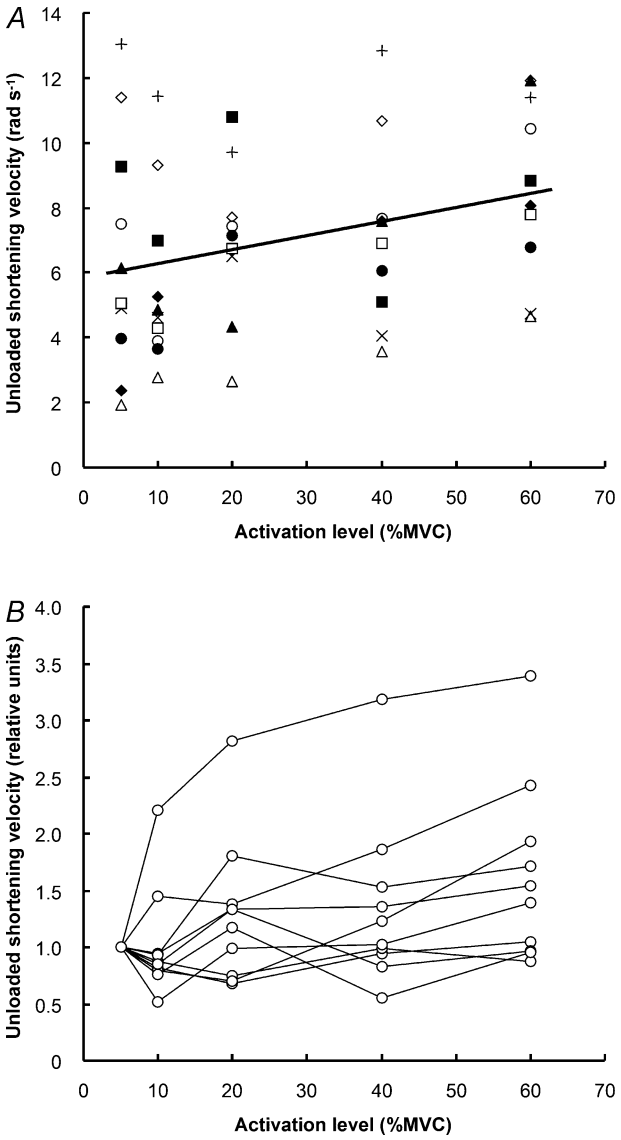

Figure 6. Unloaded shortening velocity (V_0) measured at varied activation levels (ALs)
Linear regression analysis showed that V_0 was significantly increased with AL (*A*: $n = 50$, $R^2 = 0.089$; $P = 0.035$). Different symbols represent different subjects. In addition, V_0 was expressed relative to their respective V_0 at 5%MVC to visualize the tendencies in individuals (*B*). Each line plot represents the results from each subject.

Table 1. Relations between activation level (AL) and normalized electromyographic activity (%mEMG$_{max}$) during isometric contraction just before the release (mEMG$_a$) and during shortening (mEMG$_b$)

AL (%MVC)	SOL	MG	LG	TA
mEMG$_a$				
5	10.9 ± 5.5	4.7 ± 4.0	3.8 ± 3.0	0.8 ± 1.6
10	16.9 ± 8.1	8.6 ± 6.9	6.3 ± 5.5	1.0 ± 1.2
20	25.6 ± 11.2	15.1 ± 10.7	10.8 ± 8.9	1.5 ± 1.1
40	43.5 ± 15.6	29.5 ± 17.2	23.3 ± 12.9	2.3 ± 1.1
60	62.7 ± 16.6	49.0 ± 21.3	41.1 ± 20.3	3.6 ± 1.4
mEMG$_b$				
5	16.9 ± 11.1	11.8 ± 8.5	10.8 ± 10.0	5.2 ± 4.7
10	20.0 ± 11.2	13.6 ± 8.6	10.6 ± 7.8	5.0 ± 3.3
20	25.2 ± 12.1	17.6 ± 11.2	13.7 ± 9.8	5.9 ± 3.1
40	36.3 ± 12.1	27.0 ± 14.9	21.1 ± 11.3	8.2 ± 3.5
60	50.1 ± 15.0	46.9 ± 24.7	36.5 ± 19.8	9.6 ± 5.4

Values are normalized electromyographic activity (%mEMG$_{max}$) shown as means and S.D. ($n = 10$).

maximum speed of the human triceps surae muscle. This is supported by the fact that the mean V_0 value at 60%MVC was 8.6 rad s^{-1}, which is comparable to the V_{max} of plantar flexors ranging between 6.5 and 10 rad s^{-1} in the previous studies (Hof & Van den Berg, 1981; Wickiewicz *et al.* 1984; Desplantez & Goubel, 2002; Ferri *et al.* 2003).

It cannot be denied, however, that there is a possibility for further increase in V_0 with higher AL. In isolated whole muscles, V_0 has been found to exceed V_{max}, depending on the extent of fibre heterogeneity in shortening velocity (Claflin & Faulkner, 1985, 1989; Josephson & Edman, 1988). In the human triceps surae, SOL contains a relatively high proportion of ST, whereas MG and LG appear to be heterogeneous with respect to their muscle fibre types (Johnson *et al.* 1973; Alway *et al.* 1989; Trappe *et al.* 2001). In addition, LG has the longest fascicle (fibre) lengths and the smallest fascicle (pennation) angles (Kawakami *et al.* 1998), which implies the greatest speed-generating capacity. Therefore, it is conceivable that the shortening velocity of LG primarily determines V_0 of the human triceps surae. As shown in Table 1, however, normalized activity of LG was lowest among these three muscles, and the fastest motor unit in LG might not be recruited even at 60%MVC.

We also found that there were no significant correlations between V_0 and normalized MVC (joint torque per body mass). In particular, V_0 at 60%MVC was almost independent of normalized MVC. This result suggests that at high AL (60%MVC and above) V_0 is no longer dependent on individual force-generating capacity. Given that the angular velocity is proportional to the shortening velocity of triceps surae muscle normalized with respect to its length, this is in line with the notion that the unloaded shortening velocity has no relation to the number of cross-bridges that can interact with thin filaments (Huxley, 1957).

It is worthwhile noting that the relation between V_0 and AL varied among individuals. For example, the V_0 value for subject M (Fig. 5, right panel) increased with AL, whereas that for subject K (Fig. 5, left panel) did not. This may be due to the individual differences in voluntary-activation capacity, neural reflex responses, and muscle architecture as well as errors in measurement. However, we presume that the fibre-type composition would have a significant effect. The correlation analysis showed that the correlation coefficient between normalized MVC and V_0 was higher at low ALs, although a relatively small sample size ($n = 10$) may preclude the correlations from reaching statistical significance. A tendency for positive correlation between normalized MVC and V_0 at low ALs means that the subjects with higher normalized MVC, who possibly have a higher percentage of FT, achieved higher V_0 even at low ALs. Therefore, in these subjects, FT may also constitute small motor units with low recruitment threshold and be recruited at low ALs.

Interpretation of the intercept

In the slack test with single muscle fibres, the intercept of the vertical axis by the fitted line is interpreted as a strain of SEC during isometric contraction. In the present study, a negative intercept value was frequently observed, which contradicts the traditional interpretation. This could be due to the following reasons: (1) to slacken the contracting human muscle, the finite speed of release requires a certain time during which the contractile component is supposed to shorten with a 'loaded' velocity (note that the origin of Δt axis is the onset of the release, not the onset of the slack period); (2) considerably compliant tendinous tissues of the human triceps surae (Hof, 1998; Kubo *et al.* 2002) may reduce the rate of torque redevelopment (Hill, 1951), which could cause the overestimation of Δt; and (3) the transient reduction of the agonist activity and augmentation of the antagonist activity, referred to as the unloading and stretch reflexes, respectively (Chow & Darling, 1999; Michaut *et al.* 2001; Valour & Pousson, 2003), could delay the onset of torque redevelopment. These factors might shift the relation between Δt and ΔL towards the right, and consequently make the intercept value smaller. It should be emphasized, however, that the slope of the regression line (V_0) was not considerably affected by any factors noted above, because it was determined from the 'differences' between multiple trials.

Conclusion

The present study has demonstrated that the slack test is promising for evaluating human muscle function

in vivo and its adaptability to various conditions (such as mechanical unloading, exercise training, and ageing), although the methodology still requires further refinement and validation.

References

Alway SE, MacDougall JD & Sale DG (1989). Contractile adaptations in the human triceps surae after isometric exercise. *J Appl Physiol* **66**, 2725–2732.

Arampatzis A, Morey-Klapsing G, Karamanidis K, DeMonte G, Stafilidis S & Bruggemann GP (2005). Differences between measured and resultant joint moments during isometric contractions at the ankle joint. *J Biomech* **38**, 885–892.

Chow JW & Darling WG (1999). The maximum shortening velocity of muscle should be scaled with activation. *J Appl Physiol* **86**, 1025–1031.

Claflin DR & Faulkner JA (1985). Shortening velocity extrapolated to zero load and unloaded shortening velocity of whole rat skeletal muscle. *J Physiol* **359**, 357–363.

Claflin DR & Faulkner JA (1989). The force–velocity relationship at high shortening velocities in the soleus muscle of the rat. *J Physiol* **411**, 627–637.

Claflin DR, Morgan DL & Julian FJ (1989). Effects of passive tension on unloaded shortening speed of frog single muscle fibers. *Biophys J* **56**, 967–977.

De Luca CJ, LeFever RS, McCue MP & Xenakis AP (1982). Behaviour of human motor units in different muscles during linearly varying contractions. *J Physiol* **329**, 113–128.

De Zee M & Voigt M (2001). Moment dependency of the series elastic stiffness in the human plantar flexors measured in vivo. *J Biomech* **34**, 1399–1406.

De Zee M & Voigt M (2002). Assessment of functional series elastic stiffness of human dorsiflexors with fast controlled releases. *J Appl Physiol* **93**, 324–329.

Desplantez A & Goubel F (2002). In vivo force-velocity relation of human muscle: a modelling from sinusoidal oscillation behaviour. *J Biomech* **35**, 1565–1573.

Edman KA (1979). The velocity of unloaded shortening and its relation to sarcomere length and isometric force in vertebrate muscle fibres. *J Physiol* **291**, 143–159.

Ferri A, Scaglioni G, Pousson M, Capodaglio P, Van Hoecke J & Narici MV (2003). Strength and power changes of the human plantar flexors and knee extensors in response to resistance training in old age. *Acta Physiol Scand* **177**, 69–78.

Fukunaga T, Miyatani M, Tachi M, Kouzaki M, Kawakami Y & Kanehisa H (2001). Muscle volume is a major determinant of joint torque in humans. *Acta Physiol Scand* **172**, 249–255.

Fukunaga T, Roy RR, Shellock FG, Hodgson JA & Edgerton VR (1996). Specific tension of human plantar flexors and dorsiflexors. *J Appl Physiol* **80**, 158–165.

Henneman E, Somjen G & Carpenter DO (1965). Functional significance of cell size in spinal motoneurons. *J Neurophysiol* **28**, 560–580.

Hill AV (1938). The heat of shortening and the dynamic constants of muscle. *Proc R Soc Lond B Biol Sci* **126**, 136–195.

Hill AV (1951). The effect of series compliance on the tension developed in a muscle twitch. *Proc R Soc Lond B Biol Sci* **138**, 325–329.

Hof AL (1998). In vivo measurement of the series elasticity release curve of human triceps surae muscle. *J Biomech* **31**, 793–800.

Hof AL & Van den Berg J (1981). EMG to force processing II: Estimation of parameters of the Hill muscle model for the human triceps surae by means of a calfergometer. *J Biomech* **14**, 759–770.

Huxley AF (1957). Muscle structure and theories of contraction. *Prog Biophys Biophys Chem* **7**, 255–318.

Janssen PM & De Tombe PP (1997). Protein kinase A does not alter unloaded velocity of sarcomere shortening in skinned rat cardiac trabeculae. *Am J Physiol* **273**, H2415–H2422.

Johnson MA, Polgar J, Weightman D & Appleton D (1973). Data on the distribution of fibre types in thirty-six human muscles. An autopsy study. *J Neurol Sci* **18**, 111–129.

Josephson RK & Edman KA (1988). The consequences of fibre heterogeneity on the force-velocity relation of skeletal muscle. *Acta Physiol Scand* **132**, 341–352.

Julian FJ, Rome LC, Stephenson DG & Striz S (1986). The maximum speed of shortening in living and skinned frog muscle fibres. *J Physiol* **370**, 181–199.

Kawakami Y, Ichinose Y & Fukunaga T (1998). Architectural and functional features of human triceps surae muscles during contraction. *J Appl Physiol* **85**, 398–404.

Kubo K, Kawakami Y, Kanehisa H & Fukunaga T (2002). Measurement of viscoelastic properties of tendon structures in vivo. *Scand J Med Sci Sports* **12**, 3–8.

Magnusson SP, Aagaard P, Dyhre-Poulsen P & Kjaer M (2001). Load-displacement properties of the human triceps surae aponeurosis in vivo. *J Physiol* **531**, 277–288.

Michaut A, Pousson M, Ballay Y & Van Hoecke J (2001). Short-term changes in the series elastic component after an acute eccentric exercise of the elbow flexors. *Eur J Appl Physiol* **84**, 569–574.

Minajeva A, Neagoe C, Kulke M & Linke WA (2002). Titin-based contribution to shortening velocity of rabbit skeletal myofibrils. *J Physiol* **540**, 177–188.

Muir IW, Chesworth BM & Vandervoort AA (1999). Effect of a static calf-stretching exercise on the resistive torque during passive ankle dorsiflexion in healthy subjects. *J Orthop Sports Phys Ther* **29**, 106–113; discussion 114–115.

Muraoka T, Muramatsu T, Fukunaga T & Kanehisa H (2004). Influence of tendon slack on electromechanical delay in the human medial gastrocnemius in vivo. *J Appl Physiol* **96**, 540–544.

Phillips CA & Petrofsky JS (1980). Velocity of contraction of skeletal muscle as a function of activation and fiber composition: a mathematical model. *J Biomech* **13**, 549–558.

Seger JY & Thorstensson A (2000). Muscle strength and electromyogram in boys and girls followed through puberty. *Eur J Appl Physiol* **81**, 54–61.

Sipila S, Multanen J, Kallinen M, Era P & Suominen H (1996). Effects of strength and endurance training on isometric muscle strength and walking speed in elderly women. *Acta Physiol Scand* **156**, 457–464.

Trappe SW, Trappe TA, Lee GA & Costill DL (2001). Calf muscle strength in humans. *Int J Sports Med* **22**, 186–191.

Valour D & Pousson M (2003). Compliance changes of the series elastic component of elbow flexor muscles with age in humans. *Pflugers Arch* **445**, 721–727.

Van Zandwijk JP, Bobbert MF, Harlaar J & Hof AL (1998). From twitch to tetanus for human muscle: experimental data and model predictions for m. triceps surae. *Biol Cybern* **79**, 121–130.

Vint PF, McLean SP & Harron GM (2001). Electromechanical delay in isometric actions initiated from nonresting levels. *Med Sci Sports Exerc* **33**, 978–983.

Visser JJ, Hoogkamer JE, Bobbert MF & Huijing PA (1990). Length and moment arm of human leg muscles as a function of knee and hip-joint angles. *Eur J Appl Physiol Occup Physiol* **61**, 453–460.

Wickiewicz TL, Roy RR, Powell PL, Perrine JJ & Edgerton VR (1984). Muscle architecture and force-velocity relationships in humans. *J Appl Physiol* **57**, 435–443.

Acknowledgements

We would like to thank Yoshihiko Torii for his technical support in the design and construction of the device, and Miki Onda for helpful discussion in writing the manuscript.